Ascites in cattle: ultrasonographic findings and diagnosis

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Ascites in Cattle – Ultrasonographic findings and Diagnosis

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SUMMARY

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Ultrasonography enables the examiner to detect very small amounts of fluid in the peritoneal cavity and to determine its location, amount, and sonographic features. The pathologic process responsible for the ascites, for example ileus, hepatic fibrosis, thrombosis of the caudal vena cava, or traumatic reticuloperitonitis, often can be identified. Abdominocentesis and analysis of the aspirated fluid allow differentiation of inflammatory and non-

inflammatory ascites as well as the diagnosis of uroperitoneum, hemoperitoneum, chylous ascites, and bile peritonitis.

KEYWORDS

• Cattle • Ultrasonography • Non-inflammatory ascites • Inflammatory ascites • Peritonitis • Uroperitoneum • Hemoperitoneum • Chylous ascites • Biliary ascites

Introduction

Ascites is excessive accumulation of fluid in the peritoneal cavity. Based on clinical examination alone, this condition can be difficult to diagnose because signs of abdominal fluid accumulation may be subtle and detected only after careful examination. Furthermore, the identification of the cause of ascites can be demanding and undoubtedly is facilitated by an in-depth knowledge of physiology and internal medicine. Abdominocentesis and examination of the aspirated fluid are mandatory steps in the examination of an animal with ascites. Ultrasonography allows the assessment of the extent and nature of the fluid accumulation and also identifies other pathological changes including dilation of the cranial vena cava, severe liver lesions, bladder rupture, or fibrinous deposits on abdominal organs that might point to the cause of the ascites. Ultrasonography includes evaluation of the fluid as well as the various organ systems and therefore special knowledge of the ultrasonographic features of these organs is an invaluable asset.

Diagnostic procedure in suspected cases of ascites

The diagnostic procedure for ascites includes clinical examination, urinalysis, hematological and biochemical analysis of the blood, abdominal ultrasonography, and abdominocentesis. This article focuses on the ultrasonographic examination of the abdomen.

Clinical examination

Clinical examination does not reveal ascites unless the abdominal fluid accumulation is pronounced, at which time pear-shaped enlargement of the abdomen, a strikingly flaccid abdominal wall, and sloshing of fluid upon abdominal succussion are evident. The differential diagnosis includes conditions accompanied by fluid accumulation in the gastrointestinal tract or in the pregnant uterus. Whether the fluid is intraruminal, in the intestines, uterus or the peritoneal cavity is determined during transrectal examination. Ascites accompanied by a tense abdomen, an arched back, and positive foreign body tests is suggestive of peritonitis with intraperitoneal exudate.

Blood examination

This comprises hematological and biochemical analyses including the measurement of the packed cell ~~hematocrit~~ volume and white blood cell count and the concentration of serum total solids, fibrinogen, electrolytes, urea, creatinine and bilirubin, and the activities of liver

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enzymes. The serum albumin concentration should always be determined in cattle with non-inflammatory ascites.

Ultrasonographic examination

Ultrasonography is a very sensitive, rapid, and accurate technique for the detection of peritoneal fluid.[1] Even very small amounts of fluid are readily visualized and the localization, extent, and nature of the peritoneal fluid can be assessed. In addition, it is often possible to identify the cause of the ascites.

Abdominocentesis

Abdominocentesis and examination of the aspirated fluid are required for the characterization of the ascites fluid, to differentiate non-inflammatory and inflammatory ascites, and to distinguish between uroperitoneum, hemoperitoneum, chylous ascites, and bile peritonitis.

Techniques of abdominal ultrasonography and abdominocentesis

Ultrasonography technique in suspected cases of ascites

Ultrasonographic examination is carried out on both sides of the abdomen and transrectally in the standing non-sedated animal.[2] The hair is clipped on both sides and the skin cleaned with alcohol before applying conductive gel. Linear or convex transducers with a frequency of 3.5 to 5 MHz are ideal but abnormalities close to the abdominal wall also can be assessed with a 7.5-MHz transducer. The abdomen is examined on both sides from caudal to cranial. The transducer is placed at the paralumbar fossa and then moved ventrally to the midline. The intercostal spaces 12 to 7 are also examined with the transducer held parallel to the ribs. The urinary bladder and uterus are examined transrectally with the transducer directed ventrally, and the caudal part of the left kidney is examined with the transducer directed dorsally. The abdominal organs normally occupy the entire abdominal cavity and are separated from each other and the peritoneum by capillary spaces, which contain very small amounts of serous fluid to lubricate the surface of tissues. The capillary spaces are not normally visible sonographically but can be imaged when they enlarge as a result of fluid accumulation or other disease processes. Likewise, the omentum and mesentery are difficult to visualize sonographically in healthy ruminants but are easily seen when outlined by fluid. The high fat content of these structures increases sound reflection. The organ contours usually are smooth and echoic deposits with or without fluid inclusions are considered abnormal. The fluid between organs is assessed for amount and echogenicity; the latter can range from anechoic to

106 echoic and may appear homogeneous or heterogeneous. With inflammatory ascites, echoic
107 sediment may be seen at the lowest point accompanied by a hypoechoic or even anechoic
108 supernatant. Gaseous inclusions generated by gas-producing bacteria may be seen as echoic
109 stippling. Strands of fibrin often can be seen running in a spider web-like fashion between
110 organs or between an organ and the parietal peritoneum. Normal ultrasonographic findings
111 have been published for the reticulum,[3] rumen,[4] omasum,[5] abomasum,[6] small intestines,[7]
112 colon,[8] liver,[9] spleen,[10] and urinary tract.[11–12]
113
114 *Technique for abdominocentesis in suspected cases of ascites*
115 Abdominocentesis is ideally conducted under ultrasound guidance at a site where
116 ultrasonographic changes are seen.[2] The site is clipped and the skin cleaned with alcohol and
117 disinfected. After administering local anesthesia, a 20 gauge x 3.5 in spinal needle with stylet
118 (0.90 x 90 mm, Terumo®Spinal needle, Terumo Medical Corporation, USA) is inserted into
119 the peritoneal cavity. Depending on the viscosity of the fluid, a larger-bore needle (18 gauge x
120 3.5 in, 1.20 x 90 mm) may be used. When the fluid appears heterogeneous, the collected
121 sample should include parts of the echoic sediment to increase the likelihood of obtaining
122 inflammatory or tumor cells and bacteria. The amount, color, transparency, odor, and
123 consistency of the collected fluid are assessed and the presence of cellular or other material is
124 noted (**Table 1**). A refractometer is used to measure specific gravity and total solids, and the
125 California mastitis test is used to estimate the cell count. Cytologic and bacteriologic
126 examinations are undertaken when the California mastitis test is positive. The urea and
127 creatinine concentrations are measured when uroperitoneum is suspected.
128 When assessing aspirated peritoneal fluid,[13–16] it should be remembered that the classic
129 differentiation of abdominal transudate and exudate does not always apply in sick cattle. A
130 typical exudate is defined as cloudy, watery to viscous, foul-smelling fluid that may clot
131 quickly after collection. An exudate often contains flecks of fibrin and pus. The specific
132 gravity is greater than 1.015 and the protein content greater than 30 g/L. Smears made from
133 exudates contain numerous leukocytes but the number of cells may be reduced because of
134 cytolysis. The proportion of eosinophils and neutrophils is used as a criterion for the diagnosis
135 of peritonitis;[14] fewer than 10% eosinophils and greater than 40% neutrophils is highly
136 suggestive of peritonitis. Sterile pus without bacteria may occur after antibiotic treatment of
137 the animal, and the presence of bacteria in the absence of leukocytes may indicate
138 contamination from accidental intestinal puncture. To improve the diagnostic usefulness of
139 peritoneal fluid analysis, other variables including albumin, glucose, fibrinogen, L-lactate, d-

dimers, and the activities of lactate dehydrogenase and creatine kinase were measured in serum and peritoneal fluid of 95 cows, and the peritoneal-to-serum ratios of these variables calculated.[17] The glucose concentrations of blood and peritoneal fluid are usually similar but bacteria in the peritoneal cavity metabolize glucose and cause a decrease in the peritoneal glucose concentration. The glucose concentration is therefore considered a very sensitive and specific criterion for the diagnosis of septic peritonitis in cattle.[18] L-lactate is a metabolite of anaerobic glycolysis and ischemic processes in gastrointestinal organs result in an increase in its concentration in peritoneal fluid and secondarily in blood.[18] D-dimer is a fibrin degradation product and plays an important role in the diagnosis of coagulation and thrombotic diseases. Healthy cattle have a ~~d~~D-dimer serum concentration of < 0.60 mg/L.[17] Peritonitis in cattle is associated with massive synthesis of fibrin immediately accompanied by fibrinolysis, which generates ~~d~~D-dimer. D-dimer is considered the best criterion for the diagnosis of peritonitis.[17] Analogous to other species, a sensitivity of 96 % and specificity of 98 % was calculated for D-dimer concentration in peritoneal fluid for the diagnosis of peritonitis in cattle.[18]

Kommentiert [UW3]: I think we always need to capitalize D-dimers

Types of free peritoneal fluid

There are many different types of peritoneal fluid (**Table 2**) including inflammatory, non-inflammatory, chylous, urine-like, hemorrhagic, and bilious fluids. Small fluid accumulations may only be seen at the lowest point of the abdomen but large accumulations involve the entire peritoneal cavity and the space between organs increases because of the fluid surrounding them. Because of the superb acoustic properties of fluid, the ultrasonographic visibility of organs suspended in fluid is better than the normal in-situ appearance.

Non-inflammatory ascites

Non-inflammatory ascites is the abnormal accumulation of serous fluid in the peritoneal cavity.[2] An increase in intravascular hydrostatic pressure and/or a decrease in intravascular colloid osmotic (oncotic) pressure are the principle causes of non-inflammatory ascites. It is most commonly caused by vascular congestion but can also be the result of hypoalbuminemia (decrease in oncotic pressure), retention of sodium accompanied by water retention in secondary aldosteronism, or peritoneal cancer such as mesothelioma.[19–22] Often the cause of non-inflammatory ascites is multifactorial.

Clinical signs of non-inflammatory ascites

A tentative diagnosis can be made in severe cases when symmetrical ventral abdominal enlargement causing a pear-shaped abdomen, a flaccid abdominal wall, and sloshing of fluid upon abdominal succussion are evident (**Fig. 1**). When the condition is missed or allowed to continue, the contour of the animal may progress to barrel-shaped (**Fig. 2**). Abdominal enlargement caused by ascites generally is more pronounced in calves and other young cattle than in mature cattle. However, even in cases with considerable intra-abdominal fluid accumulation, the pear-shaped abdominal appearance may be missed. Ultrasonography is the method of choice for diagnosis of ascites.

Ultrasonographic findings of non-inflammatory ascites

The typical ultrasonographic finding of non-inflammatory ascites is accumulation of anechoic fluid of varying extent in the peritoneal cavity. It may be limited to the ventral abdomen or the fluid level may extend dorsally or involve the entire peritoneal cavity. Large amounts of fluid surround the organs so that they are suspended in it. On the right side, intestines enclosed by the greater omentum are seen from the flank and intercostal spaces (**Fig. 3**). The surrounding fluid renders the greater omentum echoic and both walls of the omental bursa, each consisting of two serous layers, can often be seen (**Fig. 4**). The liver is displaced dorsally by the fluid and seen in the costal part of the abdomen. However, with mild or moderate ascites, only the ventral part of the liver including the gall bladder is surrounded by fluid (**Fig. 5**). With severe ascites, the liver is displaced from the abdominal wall creating an anechoic seam between the parietal peritoneum and the liver. Hepatic ligaments are often seen as fine echoic strands between the liver and the abdominal wall. On the left side, the rumen is displaced from the abdominal floor by anechoic fluid (**Fig. 6**). Likewise, the reticulum is displaced dorsally by fluid (**Fig. 7**). Owing to the acoustic properties of peritoneal fluid, the different layers of the reticular wall often are very distinct: the tunica serosa appears as a thin echoic line on the outside, the tunica muscularis as a thin hypoechoic line in the middle, and the tunica mucosa combined with the tela submucosa on the inside are distinct. In contrast to reticuloperitonitis, there are no signs of inflammation such as echoic fibrin deposits or abscesses. Furthermore, an anechoic seam of varying width is evident between the reticulum and spleen because the capillary space between these two organs is enlarged by the fluid.

Causes of non-inflammatory ascites

The most common causes of non-inflammatory ascites attributable to vascular congestion include chronic right-sided cardiac insufficiency, mediastinal masses, chronic liver and kidney diseases, small intestinal ileus and enteropathies, tumors of the peritoneum, and caudal vena cava thrombosis or compression (**Table 3**).

Right-sided cardiac insufficiency as the cause of ascites

Ascites caused by right-sided cardiac insufficiency (traumatic pericarditis, valvular endocarditis, idiopathic cardiomyopathy, cardiac lymphosarcoma) is accompanied by abnormal auscultatory findings such as tachycardia, pericardial or endocardial heart sounds, or cardiac arrhythmia (summation gallop heart sound in cardiomyopathy).[23] The jugular veins are distended and there is submandibular, presternal, and ventral edema. Pleural effusion, dilation of the caudal vena cava and a change in its cross-sectional appearance from triangular to oval or circular are consistent and specific ultrasonographic findings of right-sided cardiac insufficiency.[2] The activities of liver enzymes are increased because of liver congestion.

Mediastinal masses as the cause of ascites

Compression of the caudal vena cava by a mediastinal mass such as an abscess or tumor also results in a change in its cross-sectional shape to oval or circular, which can be detected sonographically in the 11th and 12th intercostal spaces. Jugular distension is not a feature of this condition.[2]

Liver disease as the cause of ascites

Ascites is common in liver disease and occurs when hepatic perfusion is compromised by liver cirrhosis, fascioliasis, or a tumor or abscess causing intrahepatic congestion. Hypoalbuminemia from impaired albumin synthesis is responsible for a decrease in intravascular oncotic pressure in liver cirrhosis and fascioliasis.[2] In addition, an increase in pressure in the venous part of the splanchnic circulation because of impaired liver perfusion causes transudation of fluid into the peritoneal space. In cattle, severe hypoalbuminemia is always accompanied clinically by edema. Prehepatic portal hypertension caused by portal vein thrombosis alone in the absence of liver disease is not associated with ascites, which underlines the crucial role of decreased oncotic pressure in the pathogenesis of ascites;[24] the oncotic pressure remains unchanged because arterial perfusion compensates for lost venous perfusion. In addition to ascites, characteristic ultrasonographic features of portal

hypertension include focal changes with abscesses or tumors, calcification of bile ducts with fascioliasis, and dilation of the portal vein. Blood analysis shows hypoalbuminemia and increased activity of liver enzymes, and microscopic examination of bile collected under ultrasound guidance shows common liver fluke ova.[25] Fascioliasis is commonly complicated by concurrent peritonitis and analysis of peritoneal fluid may show a modified transudate or even an exudate with an elevated eosinophil count. The principal ultrasonographic finding in cows with hepatic fibrosis attributable to *Senecio alpinus* poisoning was severe non-inflammatory ascites.[26] The liver parenchyma of all cows appeared heterogeneous and often had nodular changes, and all cows had portal hypertension caused by intrahepatic changes, dilation of the portal vein, and a decrease in the diameter of the caudal vena cava lumen because of reduced liver perfusion. Portal hypertension also resulted in edema of the gall bladder wall, small intestines, and greater omentum. Cows with hepatocellular carcinoma had severe focal or diffuse hepatic changes but no ascites.[2]

Kidney disease as the cause of ascites

Amyloid nephrosis is accompanied by massive protein loss in the urine resulting in hypoproteinemia, edema, and ascites.[29] The kidneys are enlarged and affected cattle have diarrhea and weight loss. Ultrasonography confirms enlargement of the kidneys and shows diffuse parenchymal changes. The histological examination of a biopsy specimen confirms the diagnosis.

Ileus as the cause of ascites

Various forms of ileus of the small and large intestines can cause ascites through transudation as a result of vascular constriction. Ileus of the small intestine includes all conditions that are accompanied by displacement, twisting and constriction of the intestine including volvulus, compression, invagination, incarceration, and obstruction.[30] Dilated, non-motile loops of small intestine that may or may not be separated from each other by fluid is a typical ultrasonographic finding of ileus (**Fig. 8**). Occasionally it is possible to visualize the cause of the ileus, for instance intestinal obstruction by blood coagula in hemorrhagic bowel syndrome (**Fig. 9**). Cecal torsion is the main disorder of the large intestine that causes transudation and peritoneal fluid accumulation (**Fig. 10**).[31,32] Ileus and cecal dilation are readily diagnosed clinically and sonographically and differentiated from other forms of ascites.[30–32]

Tumors of the peritoneum, serous membranes, and omentum

Tumors of the visceral and parietal peritoneum, and omentum are rare in cattle. They are mostly multiple, firm, nodular or pedunculated tumors or tumors with a broad attachment. Accompanying non-specific clinical signs include weight loss, poor appetite, and ascites.[2] The tumors often can be palpated transrectally. These tumors include mesothelioma,[19,21,32] metastatic hepatic, gastrointestinal, urinary tract, or pulmonary carcinoma as well as lymphoma. The differential diagnosis of peritoneal changes palpated transrectally includes extrapulmonary tuberculosis of the serous membranes and the differential diagnosis of palpable mesenteric changes includes fat necrosis. Cows with mesothelioma have severe ascites.[19-21,32] Ultrasonographically, the tumors appear as uneven nodular masses on the peritoneum and serosal surface of abdominal organs such as the rumen, omasum, or spleen (**Fig. 11**). The greater omentum, suspended in ascites fluid, may also be affected by nodular changes (**Fig. 12**). The aspirated peritoneal fluid is a modified transudate with a dark yellow to red color and may have a high erythrocyte count. The cytologic diagnosis of mesothelioma is often limited by the paucity of cells in the aspirated fluid. The final diagnosis is based on histologic, immunohistologic, or electron microscopic examination of the tumor.

Obstruction and compression of the caudal vena cava as the cause of ascites

Thrombosis and compression of the caudal vena cava by an abscess or tumor can lead to ascites. Although white thrombi of the caudal vena cava are relatively common, they are a rare cause of ascites because of the collateral circulation through the milk veins, azygos vein and spinal veins, which carry blood back to the heart when the caudal vena cava is obstructed. However, ascites occurs when the thrombus is located cranial to the liver and occupies at least half of the lumen of the vessel, [33,34] or when both milk veins are thrombosed after the infusion of irritating solutions and no longer contribute to the collateral circulation.[2] Caudal vena cava thrombosis may cause ascites in young cattle[2] and bulls because of the small diameter and capacity of the milk vein (**Fig. 13**). Obstruction or compression of the caudal vena cava leads to congestion of the vessel, which increases its diameter and changes its cross-sectional shape from triangular to oval or circular. This is best visualized in the liver region at the 11th and 12th intercostal spaces on the right side (**Fig. 14**).[35-38] As a result of liver congestion, the veins joining the congested vena cava, particularly the right hepatic vein, appear as prominent and markedly dilated vessels (**Fig. 15**), the liver has an obtuse marginal angle, and the wall of the gall bladder is edematous (**Fig. 16**). The thrombus rarely can be visualized sonographically

(**Fig. 17**) because usually it is in a section of the vena cava superimposed by the lungs.[38] Likewise, thrombi in the tributaries of the vena cava are rarely visualized.[46]

Inflammatory ascites (peritonitis)

Inflammatory ascites is associated with peritonitis, which is defined as peritoneal inflammation resulting from trauma and/or bacterial infection.[2] Peritonitis is often parasite-related and in rare cases caused by medications. Peritonitis is characterized by peritoneal alteration and proliferation as well as exudation and can be focal or diffuse and acute or chronic. Bacteria commonly involved in the etiology of peritonitis include *Trueperella pyogenes*, streptococci, staphylococci, and *Escherichia coli,* which are introduced directly into the peritoneal cavity via perforating wounds or indirectly via the hematogenous or lymphatic route. A perforating reticular foreign body is the most common cause of peritonitis (**Table 4**). Perforating abomasal ulcer also is a common cause of peritonitis in postpartum dairy cows, whereas perforating injuries of the uterus, cervix, vagina, bladder, small and large intestines, or rectum associated with parturition, urolithiasis or transrectal examination are less common. Peritonitis may also occur after laparotomy[41] or as a sequel to ulcerative colitis and proctitis.[42] Migrating juvenile stages of *Fasciola hepatica* occasionally cause peritonitis, whereas rupture of an intraperitoneal abscess is a rare cause. A tentative diagnosis of peritonitis usually is straightforward after a perforating injury of the abdominal wall associated with ruminal trocarization,[41] horn injury, or after laparotomy.

Ultrasonographic findings of focal and generalized peritonitis

The localization, extent and type of changes caused by peritonitis vary greatly depending on the severity and cause of peritonitis. Exudation may be absent in fibrinous peritonitis and the only lesions seen sonographically may be changes on the organ surface and/or peritoneum. These changes are typical of traumatic reticuloperitonitis and perforating abomasal ulcer, in which an uneven organ surface with echoic deposits (**Fig. 18**), often containing fluid inclusions (**Fig. 19**) and surrounded by fluid (**Fig. 20**), is seen instead of the normal smooth surface. The extent of the inflammatory exudation varies greatly and is usually limited to small effusions in the caudoventral region of the reticulum in cattle with hardware disease. In these cases, the reticulum, the dorsal blind sac of the rumen, and sometimes the cranial part of the ventral sac of the rumen are displaced dorsally by the effusion. Generalized peritonitis may be accompanied by massive fluid accumulation, which surrounds the organs and displaces them dorsally. Depending on the cell count and amount of fibrin, the fluid may

appear anechoic (**Fig. 21**), hypoechoic (**Fig. 22**), or echoic (**Fig. 23**). Fibrin septa that form fluid compartments (**Fig. 24**) and floating fibrin strands are often seen running in a spider web-like fashion between organs or between an organ and the parietal peritoneum. Often the fluid appears chambered in a sponge-like fashion by strands of fibrin (**Fig. 25, 26**). A layer of sediment composed of echoic cells and fibrin and an anechoic supernatant may be seen when there are many cells and a large amount of fibrin (**Fig. 24**), or abdominal organs may be coated with fibrinous deposits (**Fig. 27**) that contain gaseous inclusions of microbial origin (**Fig. 28**). When gastrointestinal contents escape into the peritoneal cavity, for instance after abomasal or intestinal rupture, echoic ingesta may be seen coating some of the organs. The intestinal walls often are thickened and echoic fibrin and inflammatory fluid are seen between loops of intestines in cattle with generalized peritonitis. Intestinal motility usually is severely reduced or absent because of adhesions, and diarrhea is common. However, based on ultrasonographic appearance alone, it is often not possible to differentiate a transudate, a modified transudate of non-inflammatory ascites, and an exudate, and the analysis of fluid collected by abdominocentesis is required for a diagnosis.

Omental bursitis

Omental bursitis is a special form of peritonitis characterized by the accumulation of watery, putrid, foul-smelling exudate in the omental bursa. Peritonitis is limited to the bursa and demarcated from the remaining abdominal cavity by a capsule of varying thickness. Reticular foreign bodies, ruminal perforation or ulcers, abomasal ulcers, and laparotomies are the most common causes.[43] Ultrasonographically, purulent omental bursitis appears as fluid accumulation of varying but often considerable size between the abdominal wall and the ventral sac of the rumen (**Fig. 29**). Extreme cases involve the entire ruminal width and length, extending from the naval to the udder, and measure more than 10 cm vertically. The ultrasonographic appearance of the fluid and the fibrin deposits vary, and the echoic leaves of the bursa sometimes can be seen. A characteristic difference between omental bursitis and generalized peritonitis is that the former is restricted to the region ventral and lateral to the rumen on the left side, whereas the latter also involves the right side of the abdomen. Furthermore, the general demeanor and appetite are more severely affect in cattle with generalized peritonitis.

Uroperitoneum

Uroperitoneum is accumulation of urine in the peritoneal cavity. A review of the causes, diagnosis, and treatment of uroperitoneum in cattle was recently published.[44] Uroperitoneum can result from leakage of urine from the kidneys, ureters, urinary bladder, and urethra[45] as well as from rupture of a persistent urachus.[46,47]

The most common cause of bladder rupture is dystocia in cows[23,48,49,50,51] and urinary obstruction[50] associated with urolithiasis or urethral compression by a hematoma[52] in male cattle. A persistent urachus may rupture spontaneously for reasons that remain unclear. This has been described in calves[53,54] and in young[55] and mature cattle.[46,47,56] The main clinical sign is pear-shaped enlargement of the abdomen.

Ultrasonography shows massive fluid accumulation involving the entire abdomen, and dorsally displaced abdominal organs that are suspended in the fluid. The site of bladder rupture can sometimes be detected during transrectal sonographic examination. The ruptured bladder may be collapsed and flaccid and contain little or no urine or contain moderate amounts of urine when the defect is sealed by fibrin.[57] When a persistent urachus exerts traction on the bladder, the urine-filled transition from the bladder to the urachus can be seen sonographically (**Fig. 30**). Sometimes the bladder is surrounded by urine. A completely empty bladder may not be seen.

The diagnosis is confirmed by abdominocentesis and analysis of the aspirated fluid. The fluid is light yellow or colorless and occasionally smells of urine. Comparison of urea and creatinine concentrations of the aspirated fluid and blood reliably differentiates urine and a transudate. With uroperitoneum, both variables are much higher in the aspirated fluid than in blood; a peritoneal fluid-to-serum creatinine concentration ratio of 2 or greater is diagnostic of uroperitoneum.[23]

A flexible endoscope is used to differentiate bladder rupture and urachal rupture.[47] With urachal rupture, the bladder is stretched longitudinally and the endoscope can be introduced into the urine-filled urachus.[47]

Hemoperitoneum

Hemoperitoneum is the presence of blood in the peritoneal cavity. It is accompanied clinically by pronounced anemia and decreased hematocrit. It is rare in cattle and always results from hemorrhage from a ruptured blood vessel or spleen or is related to a clotting disorder.[2] The ultrasonographic appearance of the fluid varies from hypoechoic to hyperechoic depending on the degree of clotting. A symptomatic diagnosis is made when abdominocentesis produces a

hemorrhagic aspirate but the measurement of clotting factors or a laparotomy may be required for an etiological diagnosis. There have been two reports of hemoperitoneum in cattle as a result of a ruptured metastatic granulosa cell tumor[59,59] and another because of rupture of the gall bladder.[60] All animals had marked anemia and the abdominal fluid was anechoic. One of the cows with metastatic granulosa cell tumor had multiple metastatic tumors in the abdominal cavity that appeared ultrasonographically as multichambered sponge-like structures of varying size (**Fig. 31**). Hemorrhage was caused by rupture of the cystic tumor capsule. Two other (unpublished) cases referred to our clinic concerned two cows with hemoperitoneum following transrectal manual rupture of an ovarian cyst and enucleation of a corpus luteum. In both cases the peritoneal fluid was hypoechoic.

Chylous ascites

Chylous ascites is the accumulation of chyle in the peritoneal cavity and arises from a blockage of the thoracic duct. Neoplasia, inflammatory processes, tuberculosis, or trauma are causes of chylous ascites in people but some cases are idiopathic.[61] This condition may also result from iatrogenic injury to lymph vessels during surgery.[62] The fluid appears hyperechoic, and aspiration of viscous, milky, opaque fluid rich in triglycerides is diagnostic. Chylothorax and chyloperitoneum in a calf diagnosed using mesenteric lymphangiography is the only published case in cattle.[63] Another case of chylothorax occurred in a two-day-old calf as a result of thoracic vertebral fracture during birth.[64]

Biliary ascites (bile peritonitis)

Biliary ascites (bile peritonitis) is the term used to describe peritonitis caused by bile in the peritoneal cavity. Possible causes are perforation of the gall bladder by trauma or because of cholecystitis or iatrogenic rupture during procedures such as aspiration of bile from the gall bladder. Only three cases of bile peritonitis have been reported in cattle and all were the result of bile duct obstruction by concrement related to *Fasciola hepatica* infestation.[60] Typical clinical signs of cholestasis including icterus and bilirubinuria were seen in only one cow. Another cow had severe anemia because gall bladder rupture was accompanied by hemoperitoneum. Increased activity of the bile duct enzyme γ-glutamyl transferase and increased concentration of bilirubin are the main serum biochemical findings. Ultrasonographic examination of cows with gall bladder rupture shows biliary ascites, fibrin deposits on various organs, and strands of fibrin in the peritoneal cavity. In addition, there are fascioliasis-related changes in the liver, which primarily include calcified echoic bile ducts

with distal acoustic shadowing and cholestasis. The appearance of the aspirated fluid varied widely in the three cows seen at our clinic and was hemorrhagic, bilious, and greenish opaque and fibrinous, respectively. In one cow, the gall bladder contained sediment and had a thickened wall and irregular contour, which was related to the rupture. The bilirubin concentration is increased in bile-containing ascites fluid[65] and should be measured when the assessment of the aspirated fluid is not straightforward. A bilirubin concentration greater than 8.5 umol/L indicates rupture of the gall bladder.[66]

Differential diagnosis of intraabdominal fluid accumulation

The differential diagnosis of intraabdominal fluid accumulation includes normal pregnancy and hydrops allantois. However, the diagnosis of pregnancy and hydrops usually is straightforward based on the results of transrectal examination. On sonograms, the uterine wall is seen as a thick echoic line and the caruncles appear as fist-sized structures attached to the uterine wall. Furthermore, fetal parts are seen in pregnant cows and a heartbeat is often detected in live fetuses.

~~**Summary**~~

~~Ultrasonography enables the examiner to detect very small amounts of fluid in the peritoneal cavity and to determine its location, amount, and sonographic features. The pathologic process responsible for the ascites, for example ileus, hepatic fibrosis, thrombosis of the caudal vena cava, or traumatic reticuloperitonitis, often can be identified. Abdominocentesis and analysis of the aspirated fluid allow differentiation of inflammatory and non-inflammatory ascites as well as the diagnosis of uroperitoneum, hemoperitoneum, chylous ascites, and bile peritonitis.~~

References

1. Schreiber MA, Kremer H. Peritonealhöhle, Aszites und Bauchdecken. In: Kremer H, Dobrinski W, editors. Sonographische Diagnostik. Innere Medizin und angrenzende Gebiete. München, Wien, Baltimore: Urban & Schwarzenberg; 1994. pp. 235-41.
2. Braun U. Atlas und Lehrbuch der Ultraschalldiagnostik beim Rind. Berlin: Parey Buchverlag; 1997.
3. Braun U, Rauch S. Ultrasonographic evaluation of reticular motility during rest, eating, rumination and stress in 30 healthy cows. Vet Rec 2008; 163 (19): 571-4.

4. Braun U, Schweizer A, Trösch L. Ultrasonography of the rumen of dairy cows. BMC Vet Res 2013; 9:44.
5. Braun U, Blessing S. Ultrasonographic examination of the omasum in 30 healthy cows. Vet Rec 2006; 159 (24): 812-5.
6. Braun U, Wild K, Guscetti F. Ultrasonographic examination of the abomasum of 50 cows. Vet Rec 1997; 140 (4): 93-8.
7. Braun U, Marmier O. Ultrasonographic examination of the small intestine of cows. Vet Rec 1995; 136 (10): 239-44.
8. Braun U, Amrein E. Ultrasonographic examination of the caecum and proximal and spiral ansa of the colon of cattle. Vet Rec 2001; 149 (2) 45-8.
9. Braun U, Gerber D. Influence of age, breed, and stage of pregnancy on hepatic ultrasonographic findings in cows. Am J Vet Res 1994; 55 (9): 1201-5.
10. Braun U, Sicher D. Ultrasonography of the spleen in 50 healthy cows. Vet J 2006; 171 (3): 513-8.
11. Braun U. Ultrasonographic examination of the right kidney in cows. Am J Vet Res 1991; 52 (12): 1933-1939.
12. Braun U. Ultrasonographic examination of the left kidney, the urinary bladder, and the urethra in cows. J Vet Med A 1993; 40 (1): 1-9.
13. Hirsch VM, Townsend HGG. Peritoneal fluid analysis in the diagnosis of abdominal disorders in cattle: A retrospective study. Can Vet J 1982; 23 (12): 348-354.
14. Wilson AD, Hirsch VM, Osborne AD. Abdominocentesis in cattle: Technique and criteria for diagnosis of peritonitis. Can Vet J 1985; 26(2): 74-80.
15. Kopcha M, Schultze AE. Peritoneal fluid. Part 1. Pathophysiology and classification of nonneoplastic effusions. Compend Contin Educ Pract Vet 1991; 13 (3): 519-25.
16. Kopcha M, Schultze AE. Peritoneal fluid. Part 2. Abdominocentesis in cattle and interpretation of nonneoplastic samples. Compend Contin Educ Pract Vet 1991; 13 (4): 703-9.
17. Wittek T, Grosche A, Locher L, Alkaassem A, Fürll M. Biochemical constituents of peritoneal fluid in cows. Vet Rec 2010; 166 (1): 15-9.
18. Wittek T, Grosche A, Locher LF, Fürll M. Diagnostic accuracy of d-dimer and other peritoneal fluid analysis measurements in dairy cows with peritonitis. J Vet Intern Med. 2010; 24 (), 1211-1217.

19. Wolfe DF, Carson RL, Hudson RS, Boosinger TR, Mysinger PW, Powe TA, Claxton MS, Angel KL. Mesothelioma in cattle: Eight cases (1970-1988). J Am Vet Med Assoc 1991; 199 (4): 486-91.
20. Milne MH, Mellor DJ, Barrett DC, Fitzpatrick JL. Observations on ascites in nine cattle. Vet Rec 2001; 148 (11): 341-4.
21. Braun U, Gerspach C, Metzger L, Ziegler-Gohm D. Ultrasonographic findings in a cow with ascites due to a mesothelioma. Vet Rec 2004; 154 (9): 272-4.
22. Braun U, Rütten M, Bleul U, Previtali P, Krüger S, Gerspach C, Geiger A, Sydler T. Biphasisches Mesotheliom bei einer Braunviehkuh: Klinische, histomorphologische, immunhistochemische und elektronenmikroskopische Befunde. Schweiz Arch Tierheilk 2012; 154 (1): 33-8.
23. Radostits OM, Gay CC, Hinchliff KW, Constable PD. Veterinary Medicine. A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. Edinburgh: Saunders Elsevier. 2007, pp. 562-3.
24. Matern S. Leber. In: Siegenthaler W (editor). Klinische Pathophysiologie, 6th edition. Stuttgart, New York: Georg Thieme; 1987. pp 864-900.
25. Braun U, Gerber D. Percutaneous ultrasound-guided cholecystocentesis in cows. Am J Vet Res 1992; 53 (7): 1079-84.
26. Braun U, Linggi T, Pospischil A. Ultrasonographic findings in three cows with chronic ragwort (Senecio alpinus) poisoning. Vet Rec 1999; 144 (5): 122-6.
27. Braun U, Caplazi P, Linggi T, Graf F. Polyglobulie infolge Leberkarzinom bei Rind und Schaf. Schweiz Arch Tierheik 1997; 139 (4): 165-71.
28. Braun U, Nuss K, Soldati G, Ossent P. Clinical and ultrasonographic findings in four cows with liver tumours. Vet Rec 2005; 157 (16): 482-4.
29. Elitok UM, Elitok B, Unver O. Renal amyloidosis in cattle with inflammatory diseases. J Vet Intern Med 2008; 22 (2): 450-5.
30. Braun U. Ultrasonography of the gastrointestinal tract in cattle. Vet Clin North Am Food Anim Pract 2009; 25 (3): 567-90.
31. Braun U, Amrein E, Koller U, Lischer C. Ultrasonographic findings in cows with dilatation, torsion and retroflexion of the caecum. Vet Rec 2002, 150 (3): 75-9.
32. Braun U, Beckmann C, Gerspach C, Hässig M, Muggli E, Knubben-Schweizer G, Nuss K. Clinical findings and treatment in cattle with caecal dilatation. BMC Vet Res 2012; 8: 75.

33. Adams OR. Hepatic changes resulting from partial ligation of the posterior vena cava in cattle. Am J Vet Res 1963; 24 (5): 557-64.
34. Selman IE, Wiseman A, Petrie L, Pirie HM, Breeze RG. A respiratory syndrome in cattle resulting from thrombosis of the posterior vena cava. Vet Rec 1974; 94 (20): 459-66.
35. Braun U, Schefer U, Gerber D, Föhn J. Ultrasonographic findings in a cow with ascites due to thrombosis of the caudal vena cava. Schweiz Arch Tierheilk 1992; 134 (5): 235-41.
36. Braun U, Schweizer G, Wehbrink D, Müller R, Hilbe M. Ultraschallbefunde bei einem Rind mit Aszites infolge Thrombose der Vena cava caudalis. Tierärztl Prax 2005; 33 (G): 389-94.
37. Braun U, Flückiger M, Feige K, Pospischil A. Diagnosis by ultrasonography of congestion of the caudal vena cava secondary to thrombosis in 12 cows. Vet Rec 2002; 150 (7): 209-13.
38. Braun U, Salis F, Gerspach C. Sonographischer Nachweis eines echogenen Thrombus in der Vena cava caudalis bei einer Kuh. Schweiz Arch Tierheilk 2003; 145 (7): 340-1.
39. Braun U, Feller B, Trachsel D, Rütten M, Augsburger H, Riond B, Sydler T. Lungenabszess, Pleuraerguss und Aszites bei einem Rind mit Vena-cava-caudalis-Thrombose. Dtsch tierärztl Wschr 2007; 114 (5): 165-70.
40. Mohamed T, Sato H, Kurosawa T, Oikawa S. Ultrasonographic localisation of thrombi in the caudal vena cava and hepatic veins in a heifer. Vet J 2004; 168 (1): 103-6.
41. Braun U, Pusterla N, Anliker H. Ultrasonographic findings in three cows with peritonitis in the left flank region. Vet Rec 1998; 142 (13): 338-40.
42. Braun U, Hilbe M, Gerspach C, Ruetten M. Ulzerierende Colitis und Proktitis bei zwei Braunviehkühen. Schweiz Arch Tierheilk 2015; 157: 204-8.
43. Dirksen G. Bauchhöhlenabszesse, Netzbeutelentzündung. In: Dirksen G, Gründer HD, Stöber M, editors. Innere Medizin und Chirurgie des Rindes. Berlin: Parey Buchverlag. 2002, pp. 671-4.
44. Braun U, Nuss K. Uroperitoneum in cattle: Ultrasonographic findings, diagnosis and treatment. Acta Vet Scand., submitted for publication.
45. Maxie MG, Newman SJ. Acquired anatomic variations. In: Maxie MG (editor). Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, Volume 2. 5th edn. Edinburgh: Saunders Elsevier; 2007. pp 506-7.
46. Braun U, Nuss K, Wapf P, Lischer C. Clinical and ultrasonographic findings in five cows with a ruptured urachal remnant. Vet Rec 2006; 159 (23): 780-2.

47. Braun U, Previtali M, Fürst A, Wehrli M, Muggli E. Zystoskopie bei einem Rind mit Urachus persistens-Ruptur. Schweiz Arch Tierheilk 2009; 151 (11): 539-44.
48. Smith JA, Divers TJ, Lamp TM. Ruptured urinary bladder in a post-parturient cow. Cornell Vet 1983; 73 (1): 3-12.
49. Carr EA, Schott HC, Barrington GM, Parish SM. Ruptured urinary bladder after dystocia in a cow. J Am Vet Med Assoc 1993; 202 (4), 631-2.
50. Gründer HD. Krankheiten von Harnleiter, Harnblase und Harnröhre. In: Dirksen G, Gründer HD, Stöber M, editors. Innere Medizin und Chirurgie des Rindes. Berlin: Parey Buchverlag. 2002, pp. 719-36.
51. Braun U, Wetli U, Bryce B, Tschuor A, Wirz M, Wehrli Eser M. Clinical, ultrasonographic and endoscopic findings in a cow with bladder rupture caused by suppurative necrotising cystitis. Vet Rec 2007; 161 (20): 700-2.
52. Braun U, Trösch L, Sydler T. Ruptured urinary bladder attributable to urethral compression by a haematoma after vertebral fracture in a bull. Acta Vet Scand 2014; 56: 17.
53. Bell GJC, Macrae AI, Milne EM, Scott PR. Extensive uroperitoneum and pleural effusion associated with necrotic urachal remnant in a bull calf. Vet Rec 2004; 154 (16): 508-9.
54. Nikahval B, Khafi MSA. Congenital persistent urachus, urethral obstruction and uroperitoneum in a calf. Iran J Vet Res 2013; 14 (2): 158-60.
55. Baxter GM, Zamos DT, Mueller POE. Uroperitoneum attributable to ruptured urachus in a yearling bull. J Am Vet Med Assoc 1992; 200 (4): 517-20.
56. Marques LC, Marques JA, Marques ICS, Teixeira MCA. Dilatação cística do úraco e uroperitônio em touros: relato de cinco casos. Arq Bras Med Vet Zootec 2010; 62 (6): 1320-4.
57. Floeck M. Ultrasonography of bovine urinary tract disorders. Vet Clin North Am Food Anim Pract 2009; 25 (3): 651-67.
58. Masseau I, Fecteau G, Desrochers A, Francoz D, Lanthier I, Vaillancourt D. Hemoperitoneum caused by the rupture of a granulosa cell tumor in a Holstein heifer. Can Vet J 2004; 45 (6): 504-6.
59. Trösch L, Müller K, Brosinski K, Braun U. Hämoabdomen und Hämothorax bei einem Rind mit metastasierendem Granulosazelltumor. Schweiz Arch Tierheilk 2015, ~~157~~ 157 (6), 345-7. (...), ~~in press.~~
60. Braun U, Schweizer G, Pospischil A. Clinical and ultrasonographic findings in three cows with ruptured gallbladders. Vet Rec 2005; 156 (11): 351-3.

Kommentiert [UW4]: To be filled soon I presume

61. Siegenthaler W. Allgemeine Differenzialdiagnose des Ikterus. In: Siegenthalers Differenzialdiagnose. Innere Krankheiten – vom Symptom zur Diagnose. Stuttgart: Georg Thieme Verlag. 2000, pp. 773-809.
62. Lee EW, Shin JH, Ko HK, Park J, Kim SH, Sung KB. Lymphangiography to treat postoperative lymphatic leakage: A technical review. Korean J Radiol 2014; 15 (6): 724-32.
63. Cruz AM, Riley CB, MacDonald DG, Ferguson JG. Use of mesenteric lymphangiography in a calf with chylothorax and chyloperitoneum. J Am Vet Med Assoc 1995; 206 (10): 1567-71.
64. Pusterla N, Pusterla JB, Thür B, Rüsch P. Chylothorax bei einem Kalb. Tierärztl Prax 1996; 24 (6): 554-8.
65. Argyres MI, Porter J, Rizeq MN. Diagnosis of clinically unsuspected gallbladder rupture by peritoneal fluid cytology - A case report. Acta Cytologica 1998; 42 (4): 973-7.
66. Dirksen G. Normalbefunde und wichtigste Abweichungen des Bauchhöhlenpunktates beim Rind. In: Dirksen G, Gründer HD, Stöber M, editors. Die klinische Untersuchung des Rindes. Berlin: Paul Parey. 1990, pp. 392-3.

Legend to Figures

Figure 1: Pear-shaped abdomen in a cow with ascites due to uroperitoneum.

Figure 2: Barrel-shaped abdomen in a cow with ascites due to uroperitoneum.

Figure 3: Ultrasonogram of ascites in a cow with compression of the caudal vena cava by a liver abscess, imaged from the right flank. Loops of small intestine are displaced dorsally by anechoic ascites fluid. 1 Lateral abdominal wall, 2 Extraomental ascites fluid, 3 Greater omentum, 4 Intraomental ascites fluid, 5 Small intestine, Ds Dorsal, Vt Ventral.

Kommentiert [UW5]: Not sure if we need to put extra-omental rather than extraomental but we'll see with the editorial team

Figure 4: Ultrasonogram of ascites in a 1.5-year-old Brown Swiss heifer with thrombosis of the caudal vena, imaged from the right flank. Loops of small intestine are displaced dorsally by hypoechoic ascites fluid. 1 Lateral abdominal wall, 2 Extraomental ascites fluid, 3 Large omentum, 4 Intraomental ascites fluid, 5 Small intestine, Ds Dorsal, Vt Ventral. See also Figure 13.

Figure 5: Ultrasonogram of the liver and gallbladder of a cow with ascites, imaged from the 10th intercostal space on the right side. The liver and gall-bladder are displaced medially by anechoic ascites fluid. 1 Lateral abdominal wall, 2 Ascites, 3 Liver, 4 Gallbladder, Ds Dorsal, Vt Ventral.

Figure 6: Ultrasonogram of ascites in a cow, imaged from the 10 intercostal space on the left side. The rumen is displaced dorsomedially by anechoic ascites fluid. 1 Lateral abdominal wall, 2 Ascites fluid, 3 Rumen, Ds Dorsal, Vt Ventral.

Figure 7: Ultrasonogram of the reticular region of a cow with ascites, imaged from the sternal part of the ventral abdomen. The reticulum is displaced dorsally by hypoechoic ascites fluid. 1 Ventral abdominal wall, 2 Musculophrenic vein, 3 Diaphragm, 4 Ascites fluid, 5 Reticulum, Cr Cranial, Cd Caudal.

Figure 8: Ultrasonogram of the abdomen of a cow with ileus because of intussusception, imaged from the 12th intercostal space on the right side. There is accumulation of anechoic fluid (transudate) and dilated loops of small intestine. 1 Abdominal wall, 2 Dilated loops of small intestine, 3 Anechoic fluid between the loops of small intestine, Ds Dorsal, Vt Ventral.

Figure 9: Ultrasonogram of the abdomen of a cow with hemorrhagic bowel syndrome, imaged from the right flank. Anechoic fluid (transudate) and dilated loops of small intestine. Small intestine contain echoic material surrounded by an anechoic rim consistent with a blood clot. 1 Abdominal wall, 2 Anechoic fluid between loops of small intestine, 3 Small intestine with a blood clot, Ds Dorsal, Vt Ventral.

Figure 10: Ultrasonogram of the abdomen of a cow with dilation and retroflexion of the cecum, imaged from the right flank. The cecum and colon are separated by anechoic fluid (transudate). 1 Lateral abdominal wall, 2 Fluid accumulation, 3 Cecum, 4 Colon, Ds Dorsal, Vt Ventral.

Figure 11: Ultrasonogram (A) and postmortem view (B) of the spleen of a cow with mesothelioma, imaged from the 10th intercostal space on the left side. The spleen is covered with yellow tumorous nodules identified histologically as mesothelioma. 1 Lateral abdominal wall, 2 Ascites. 3 Spleen, 4 Tumorous alterations of the spleen (mesothelioma), 5 Rumen, Ds Dorsal, Vt Ventral.

Figure 12: Ultrasonogram of ascites in a cow with mesothelioma showing nodular changes of the greater omentum, imaged from the right flank. 1 Lateral abdominal wall, 2 Ascites, 3 Greater omentum, 4 Tumorous nodules (mesothelioma), Ds Dorsal, Vt Ventral.

Figure 13: Pear-shaped abdomen in a Brown-Swiss-heifer with ascites as a sequel of thrombosis of the caudal vena cava. See also Figure 4.

Figure 14: Ultrasonogram of the liver in a cow with thrombosis of the caudal vena cava, imaged from the 11th intercostal space on the right side. The caudal vena cava, which normally is triangular in cross-section, has become oval in cross-section because of congestion and dilation. 1 Lateral abdominal wall, 2 Liver, 3 Dilated caudal vena cava with oval cross-section and a large diameter of 6.24 cm, Ds Dorsal, Vt Ventral.

Figure 15: Ultrasonogram of the liver in a cow with traumatic pericarditis, imaged from the 11th intercostal space on the right side. The caudal vena cava and right hepatic vein are

dilated. 1 Lateral abdominal wall, 2 Liver, 3 Dilated caudal vena cava with oval cross-section, 4 Dilated right hepatic vein, 5 Portal vein, Ds Dorsal, Vt Ventral.

Figure 16: Ultrasonogram of the liver in a Brown-Swiss heifer with ascites caused by thrombosis of the caudal vena cava, imaged from the 10th intercostal space on the right side. The liver has an obtuse marginal angle and the wall of the gallbladder is edematous. 1 Abdominal wall, 2 Ascites fluid, 3 Liver, 4 Gallbladder, Ds Dorsal, Vt Ventral, see also Figures 4 and 13.

Figure 17: Ultrasonogram of the liver of a cow with caudal vena cava thrombosis, imaged from the 11th intercostal space on the right side. The caudal vena cava is occupied by an echoic thrombus. 1 Abdominal wall, 2 Liver, 3 Echoic thrombus in the caudal vena cava, 4 Portal vein, Ds Dorsal, Vt Ventral.

Figure 18: Ultrasonogram of the reticulum in a cow with traumatic reticuloperitonitis, imaged from the sternal part of the abdomen. There are massive fibrin deposits caudal to the reticulum. 1 Abdominal wall, 2 Reticulum, 3 Echoic fibrin deposits, Cr Cranial, Cd Caudal.

Figure 19: Ultrasonogram of the reticular region in a cow with traumatic reticuloperitonitis, imaged from the sternal part of the abdomen. There are massive fibrin deposits and some fluid pockets between the reticulum and the rumen. 1 Abdominal wall, 2 Musculophrenic vein, 3 Diaphragm, 4 Reticulum, 5 Echoic deposits with fluid pockets, 6 Anterior dorsal blind sac of the rumen, Cr Cranial, Cd Caudal.

Figure 20: Ultrasonogram of the reticular region of a cow, imaged from the sternal part of the abdomen. An effusion and fibrin deposits are evident caudal to the reticulum. 1 Abdominal wall, 2 Musculophrenic vein, 3 Diaphragm, 4 Reticulum, 5 Effusion, 6 Fibrin deposits.

Figure 21: Ultrasonogram of ascites in a cow with type 4 abomasal ulcer, imaged from the ventral part of the right abdomen. The ascites fluid is anechoic. 1 Ventral abdominal wall, 2 Ascites fluid, 3 Rumen, Cr Cranial, Cd Caudal.

Figure 22: Ultrasonogram of ascites in a cow with generalized peritonitis, imaged from the ventral abdominal wall. The ascites fluid is hypoechoic. 1 Ventral abdominal wall, 2 Hypoechoic ascites fluid, 3 Fibrin strand, Cr Cranial, Cd Caudal.

Figure 23: Ultrasonogram of ascites in a cow with generalized peritonitis, imaged from the ventral abdominal wall. The ascites fluid is echoic. 1 Ventral abdominal wall, 2 Echoic ascites fluid, 3 Fibrin deposits, Cr Cranial, Cd Caudal.

Figure 24: Ultrasonogram of ascites in a cow with generalized peritonitis caused by type 4 abomasal ulcer, imaged from the ventral abdominal wall. The hypoechoic ascites fluid is separated by strands of fibrin. 1 Ventral abdominal wall, 2 Sediment on the floor of the abdomen, 3 Fluid accumulation, 4 Strands of fibrin, Cr Cranial, Cd Caudal.

Figure 25: Ultrasonogram of the abdomen of a cow with generalized peritonitis caused by a perforating foreign body in the abomasum, imaged from the ventral abdominal wall. The peritoneal fluid is separated into chambers by strands of fibrin, giving it a sponge-like appearance. 1 Ventral abdominal wall, 2 Multichambered fibrin network. Cr Cranial, Cd Caudal.

Figure 26:Ultrasonogram of the abdomen of a cow with generalized peritonitis caused by perforation of the uterus, imaged from the ventral abdominal wall. The peritoneal fluid is separated into chambers by strands of fibrin, giving it a sponge-like appearance . 1 Ventral abdominal wall, 2 Multichambered fibrin network, 3 Greater omentum. Cr Cranial, Cd Caudal.

Figure 27: Ultrasonogram of the abdomen of a cow with generalized peritonitis caused by a perforating reticular foreign body, imaged from the ventral abdominal wall. The ventral sac of the rumen is covered by a thick fibrin layer. 1 Ventral abdominal wall, 2 Anechoic fluid, 3 Fibrin deposits on the ventral aspect of the ruminal wall, 4 Rumen, Cr Cranial, Cd Caudal.

Figure 28: Ultrasonogram of the abdomen of a cow with omental bursitis caused by type 4 abomasal ulcer, imaged from the 10th intercostal space on the left side. The fluid in the omental bursa is characterized by echoic stippling indicative of microbial gas production. 1

Lateral abdominal wall, 2 Fluid in the omental bursa with echoic stippling, 3 Ruminal wall, Ds Dorsal, Vt Ventral.

Figure 29: Ultrasonogram of the abdomen of a cow with omental bursitis caused by type 4 abomasal ulcer, imaged from the ventral abdominal wall. There is echoic stippling of the fluid in the omental bursa indicative of microbial gas production. 1 Ventral abdominal wall, 2 Greater omentum, 3 Fluid in the omental bursa with echoic stippling, 4 Gas inclusions, Cr Cranial, Cd Caudal.

Figure 30: Ultrasonogram of the bladder obtained transrectally in a cow with rupture of a persistent urachus. The persistent urachus is visible at the cranial pole of the bladder. 1 Rectum, 2 Anechoic fluid (uroperitoneum) surrounding the bladder. 3 Bladder, 4 Persistent urachus, Cr Cranial, Cd Caudal.

Figure 31: Ultrasonogram of the abdomen of a cow with hemoabdomen caused by rupture of the capsule of a granulosa cell tumor, imaged from the right flank. Metastatic tumors are seen on the surface of internal organs. 1 Abdominal wall, 2 Anechoic fluid, 3 Metastatic tumors, Cr Cranial, Cd Caudal.

Figure 32: Ultrasonogram of the abdomen of a cow with hemoabdomen caused by rupture of the capsule of a granulosa cell tumor, imaged from the right abdominal wall. Metastatic tumors are seen on the surface of internal organs. 1 Ventral abdominal wall, 2 Anechoic fluid, 3 Metastatic tumors, Cr Cranial, Cd Caudal.

Table 1

Goals of abdominocentesis

1. Assessment of amount, color, odor, and consistency of a sample (e.g. serous, bloody, purulent, urine-like, bile-like)
2. Determination of specific gravity and concentration of total protein (total solids) using a refractometer
3. California mastitis test for semiquantitative determination of cellular content of fluid
4. Cytological, bacteriological and possibly biochemical evaluation of fluid
5. Comparison of urea and creatinine concentrations of peritoneal fluid and serum

Table 2

Types and characteristics of intraabdominal peritoneal fluid accumulation

Type of intraabdominal fluid	Characteristics
Inflammatory ascites	Exudate
Non-inflammatory ascites	Transudate or modified transudate
Chylous ascites	Milky fluid
Uroperitoneum	Urine-like fluid
Haemoperitoneum	Bloody fluid
Biliary ascites	Bilious fluid

Table 3

Non-inflammatory ascites in cattle: causes and ultrasonographic, clinical and laboratory findings

Affected organ	Disease	Main clinical and laboratory findings	Ultrasonographic findings
Heart	Right-sided cardiac insufficiency	1) Abnormal auscultatory findings, such as tachycardia, murmur, ~~arrhythmia~~dysrrhythmia 2) Distension of jugular veins 3) Oedema 4) Elevated activities of liver enzymes	1) Abnormal echocardiographic findings 2) Pleural effusion 3) Dilatation of caudal vena cava
Mediastinum	Mediastinal mass, such as a tumour or abscess	1) Radiodensity on thoracic radiograph 2) Elevated activities of liver enzymes	Dilatation of caudal vena cava
Liver	Liver cirrhosis Fascioliasis Liver abscess Liver tumour	1) Elevated activities of liver enzymes 2) Liver fluke eggs in faeces or bile (fascioliasis) 3) Shorter-than-normal glutaraldehyde clotting test (liver abscess)	1) Dilatation of portal vein (liver cirrhosis) 2) Calcification of bile ducts (fascioliasis) 3) Discrete changes in liver parenchyma (abscess, tumour)
Kidneys	Nephrotic syndrome Amyloidnephrosis	1) Oedema 2) Enlarged left kidney on rectal examination 3) Diarrhea 4) Massive proteinuria 5) Elevated level of serum urea	Enlargement of kidneys; renal parenchyma diffusely echogenic in amyloidnephrosis
Intestine	Volvulus Enteropathy	1) Signs of ileus (volvulus) 2) Weight loss, diarrhea (enteropathy)	Jejunum dilated and static
Peritoneum	Tumours, such as mesothelioma	No specific findings	1) Neoplastic changes of peritoneum, omentum and serosal surfaces 2) Metastatic tumors in other organs (not in mesothelioma)
Blood vessels	Thrombosis of caudal vena cava	1) Metastatic suppurative bronchopneumonia 2) Epistaxis 3) Elevated activities of liver enzymes	1) Dilatation of caudal vena cava 2) Thrombus in caudal vena cava (sometimes)

Table 4

Causes of peritonitis in cattle

Organ	Cause	Nature of disease
Reticulum	Foreign body	Localized or generalized peritonitis
Abomasum	Type-3 abomasal ulcer	Localized peritonitis
	Type-4 abomasal ulcer	Generalized peritonitis
Small/large intestine	Ileus	Generalized peritonitis
	Intestinal ulcer	Generalized peritonitis
	Intestinal tumor	Generalized peritonitis
	Migrating *Fasciola hepatica* larvae	Fibrinous peritonitis
Rectum	Injury during rectal palpation	Localized or generalized peritonitis
Uterus	Injury during parturition	Localized or generalized peritonitis
	Injury during intrauterine infusion	Localized or generalized peritonitis
Vagina	Sadism	Localized or generalized peritonitis
Urinary bladder	Injury during catheterization (females)	Localized or generalized peritonitis, and uroperitoneum after rupture of urinary bladder
	Rupture caused by obstructive urolithiasis (males)	
Abdominal wall	Trocarization of rumen	Localized or generalized peritonitis, involving ingesta after trocarization of rumen
	Laparotomy	
	Perforating injury (e.g. injury caused by horns)	
All abdominal organs	Rupture of abscess	Localized or generalized peritonitis
Systemic infection	Pasteurellosis, tuberculosis, Anthrax	Generalized peritonitis